The fortunate termination of previous labors by no means does away with the necessity for this examination. It is an undoubted fact, amply sustained by experience, that in cases of contraction the difficulties tend to increase with the number of labors, and while the first and second labors may end favorably, later ones may require the severest operative procedures.[1]

2. The examination should consist in measurement of the external conjugate, anterior and posterior iliac spines, and iliac crests. If these external measurements indicate a normal pelvis, the examination may end with them. But if contraction is suspected, the diagonal conjugate and the oblique ascending diameter of Löhlein should also be taken. These measurements should be considered in the light thrown upon them by the previous history and general condition of the patient, and especially by a careful internal and external palpation of the pelvic bones, which will reveal the necessity for further measurements if it exists.

ON THE DISTURBANCES OF THE TACTILE SENSORY FUNCTION OF THE SKIN IN CASES OF PERIPHERAL NEURITIS,

WITH THE NARRATIVES OF TWO UNUSUAL CASES OF PERIPHERAL PALSY.

By Alfred G. Barrs, M.D. (Edin.), M.R.C.P. (Lond.),
SENIOR ASSISTANT PHYSICIAN TO THE GENERAL INFIRMARY AT LEEDS; LECTURER ON PHYSIOLOGY
IN THE YORKSHIRE COLLEGE OF THE VICTORIA UNIVERSITY

The disturbances of the tactile sensory function of the skin, in their extent, distribution, and quality, are apparently, in the experience of all observers, the most uncertain and variable of the manifestations of multiple peripheral neuritis—so much so, that now and again it would appear as though such disturbances were more accidental than essential in the clinical examples of that disorder which come before us.

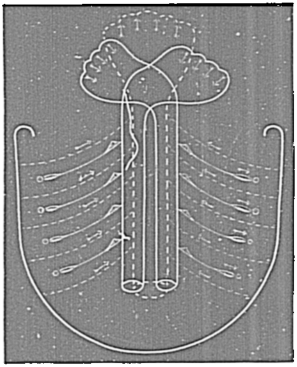
In peripheral neuritis, as in most other disorders of the nervous apparatus, it is the affection of motility which not only, as indeed is natural, most impresses the sufferer, but which is also more dwelt upon by the observer, by reason of its easy estimation, and as demanding, more directly, measures for its alleviation. The close association of sensory with motor paths in all parts of the nervous system, and a deeply rooted notion that inflammatory processes, especially those of an acute nature, are more prone to conform in their distribution to the limits of vascular and morphological areas, than to those of functional or physiological areas, have made it difficult for some to conceive of an inflammatory lesion which, in the periphery of the nervous apparatus, would pick out motor as distinct from sensory paths, or which might involve one or the other as distinguished from both.

---

[1] Michaelis: Das enge Becken, p. 195.

The self-contained nature, so to speak, of the two systems, sensory and motor, in regard to their function and disease, in spite of their close association in a morphological sense—though generally recognized, so far as the brain and spinal cord are concerned, has not been admitted to obtain in the periphery. In the spinal cord, for instance, no one hesitates to admit the occurrence of lesions distinctly and exactly confined to physiological, as distinguished from morphological, areas, where the spread of a lesion is determined and its limits defined solely by the path of some special impulse. Take, for example, the case of poliomyelitis anterior acuta, the stated lesion of infantile palsy, in which we, without much hesitation, assert that an acute inflammatory process is limited to a certain portion of the gray matter of quite arbitrary anatomical limits, while the contiguous and, anatomically speaking, continuous parts within the cord, are unaffected. The self-contained nature of the two nervous systems, efferent or motor, and afferent or sensory, is shown in the above diagram.

FIG. 1.



In this diagram, which is based upon one figured in Dr. Pye-Smith's *Syllabus of Lectures on Physiology* (London, 1885), the motor apparatus, indicated by the continuous line, is shown to consist of certain parts of the convolutions of the various arbitrary anatomical regions of the brain and spinal cord, of the peripheral nerves, muscles, tendons, and bones. The sensory apparatus, indicated by the interrupted line, consists of the general surface of the body and all the various parts leading up to and terminating in some, at present, undetermined parts of the cerebral cortex.

In looking along the whole length, so to speak, of the motor machine, it is clear that, at the two extremities, the anatomical arrangements are such as to constitute a considerable separation in locality between the tactile sensory and the motor paths. The separation has been amply established by clinical and experimental observation, as well in the spinal cord as in the brain itself, and, looking to the distal extremity, the separation in position of the ultimate ramifications of the spinal nerves is necessarily as obvious as the morphological individuality of the skin and muscle to which they are supplied. It is only when one comes to the intermediate sections of the two systems that one fails to realize so clearly their independence.

The histological identity of the two systems and their juxtaposition anatomically make it at first sight difficult to admit that in a compound nerve trunk there may be a lesion affecting alone the sensory or motor path.

Looking again at the two machines, sensory and motor, it is clear that a lesion at any point in the motor apparatus, in the brain, the spinal cord, the motor nerves, or the muscles, must cause an impairment or loss of motion in the same way that a lesion of the skin or at any point in the sensory system up to and including the tactile sensory centres, wherever they may be, must cause an alteration, loss, or impairment of tactile sensation.

My object, indeed, is to suggest that, in the consideration of the whole question of peripheral affections of the nervous system, we have two peripheries to deal with; that while both are, as far as experience at present goes, most commonly affected together, yet they may be and are affected quite independently of each other, that the systemic disposition of lesions, so readily admitted in the case of the spinal cord, must be allowed to obtain in the case of the peripheral nerves, so that lesions in the periphery may be, as in the centres, purely motor, or purely sensory, or mixed in their manifestations.

The practical point I wish to make is, that it is possible to have an extensive and pronounced affection of the peripheral motor nerves without any affection whatever of their sensory companions. The antecedent probability of such a condition is, to my own mind, clear, and of its clinical and pathological actuality I hope to be able to produce some evidence.

CASE I. *Spontaneous multiple neuritis with enlargement of the spleen, occurring in an anæmic girl; no affection of the tactile sense.*—Emily K., aged eighteen years, was admitted into the General Infirmary at Leeds under my care on March 30, 1887, complaining of general and intense muscular weakness.

History. The patient was quite well until July, 1886—that is, nine months before the clinical report here reproduced was written by Dr.

Griffith, then resident medical officer. The onset of the illness seems to have been somewhat rapid and definite, but the only circumstance which could be, in any reason, suggested as a cause, was that, on a certain day, feeling much heated, she put her feet into cold water to cool herself. After this she vomited. There was marked general disturbance, and she was confined to bed for a fortnight, during which time there was very little change in her condition. In a short time she was unable to walk, but when the weakness actually came on is not certain, as her apparently rather acute illness covered its advent. She gradually got worse, and at last was unable to move her arms or legs with any degree of force.

During the three months immediately preceding her admission she has been improving, and latterly has begun to walk again. There has not been any difficulty in swallowing, nor alteration of voice, nor evidence of laryngeal affection. There has been no scarlet fever nor any other acute illness. Menstruation was normal until the beginning of her illness, but since then she has menstruated only three times.

Present condition. Patient is an intelligent girl, of distinctly chlorotic aspect. The face is pale, and there is a slight fulness with pigmentation underneath the eyes. The lips are of fair color. She makes no complaint, except of the weakness of the limbs.

Muscular system. The patient can walk with assistance. This she does in a very slow and feeble, but not tremulous manner, It is particularly noticed that the toe is the last part of the foot to leave the ground in progression, and, looked at from behind, the soles of the feet are seen more than normally during walking. The legs and thighs are ill-nourished, and the muscles are flabby when she lies upon her back in bed. The dropped position of the foot is very striking. Both feet are extended, so that the dorsum of the foot forms an almost continuous line with the front of the leg. The great toe is not drawn down toward the sole, but is in line with the dorsum of the foot. The anterior tibial muscles are much wasted, as shown by the marked excavation between the tibia and fibula. She can raise the legs as a whole against only a moderate amount of resistance, the muscles of the thigh standing out fairly well, though they are clearly much diminished in bulk. She can extend the feet with some slight force, but cannot flex them to the smallest degree. The muscles of the calf, thigh, and gluteal region present nothing abnormal beyond general flabbiness. She can raise herself to the sitting posture, but cannot do so quickly. The spinal muscles do not appear to be seriously affected.

The upper extremities are small, the muscles flabby and of small size. She can raise her arms from her side, but not with any degree of force. There is some deficiency of the lower part of the deltoids. The latissimi and serrati appear to be unaffected. She can flex and extend the elbow-joint, and when this is done with great effort the supinator longus forms a narrow band just outside the biceps, as if its anterior part were less wasted than its posterior. The forearms have the appearance due to a very general and extreme effacement of their muscles. The hands are in a very striking condition, and manifest a rather strong suggestion of the "main en griffe" due to the prominence of the heads of the metacarpal bones and the wasting of the interossei and muscles of the palm, together with a very moderate degree of contracture of the paralyzed extensors and flexors. There is marked dropping of the wrist and for practical purposes the hands are completely useless, all attempts at using them ending in a fumbling, flail-like movement of the wrist-joint only.

In all the affected parts the most careful and repeated testing failed to find even in the smallest degree any affection whatever of tactile sensory function, and this appears to have been the case from the very beginning. She has never experienced any pain or perversion of sensation whatever.

There is occasionally the faintest response to percussion of the ligamentum patellæ on the left side, but the knee-jerk cannot be obtained on the right side. The plantaris reflex is entirely absent. The abdominal reflexes are present. The faradic reaction is seriously diminished in all the affected parts, but it cannot be said that it is, at any part, totally in abeyance.

The digestive and respiratory organs appear to be normal. There is a systolic bruit at the base of the heart, probably of pulmonary origin, and of a functional character. The spleen is distinctly enlarged, being easily felt on deep inspirations at a point three inches below the left costal margin. The liver cannot be felt. The urine is normal. Temperature 99° F. The optic disks are pale, but show no sign of disease.

The patient was of an exceedingly wayward temperament, and much to the disappointment of myself and her friends insisted upon leaving the hospital at the end of six days. However, in November, 1887, I had an opportunity of seeing her, and found her to be able to walk very much more easily than previously, though I could not satisfy myself that any substantial improvement had taken place in the condition of the upper limbs.

The symmetrical distribution of the paralysis, its exaggeration in the most distant parts of the limbs, as evidenced by the marked dropping of the feet and wrists, while the muscles of the trunk were exempt, the absence of any affection of the bladder or rectum, and the non-occurrence of bed-sores, seem to me to point directly to the peripheral origin of the disorder. The enlargement of the spleen we were unable to explain, unless it was the remainder of some acute change occurring in the onset of the illness—which I very much doubt. It did occur to me that it might be associated with the general and profound anæmia which existed.

CASE II. *Multiple peripheral neuritis following a septic wound; practically no affection of sensation.*—John M., aged thirty-eight years, an engine-tender, was sent to me by his medical attendant, who, having some suspicion that the man was suffering from an anomalous form of nervous disease, wished me to express an opinion upon the case.

The narrative of events up to January 16, 1888, when I saw him for the first time, is quite clear and definite. In the first week of October, 1887, he trapped the little finger of his left hand, and a few days later the finger, having been poulticed, was incised, and some thin watery pus set free. There was particularly well-marked lymphatic inflammation of the forearm, the skin presenting a strikingly arborescent appearance. There were swelling, pain, and hardness of the left axillary lymphatic glands. At the end of fourteen days he had practically recovered from the surgical condition, but the left hand and arm remained stiff for, perhaps, the whole of the November following.

On December 22, 1887, he returned to his medical attendant, complaining of "pins and needles" in all the limbs, with loss of grasping power, weakness of the legs, and numbness of the fingers, symptoms which he, himself, ascribed to the injury.

On January 10, 1888, thirteen weeks from the date of the injury, I saw him for the first time. He told us on this day that the stiffness of the left forearm and hand continued for three weeks after the injury, and then there appeared numbness in the fourth and fifth fingers of the left hand. Five weeks afterward the feet became numb, and were the seat of pricking sensations, and, two weeks later, the right hand was similarly affected, so that, at the end of seven weeks, the hands and feet were in a similar condition, so far as "numbness and pricking" were concerned. At the same time there was marked motor weakness in all four limbs, and the legs were slightly stiff as if from cramp. The feet were described as feeling loose at the ankle, and as scraping along the ground when walking. The gait was quite characteristic of the weakness of the anterior tibial muscles. On account of the difficulty in raising the point of the toe from the ground the whole foot had to be lifted in such a manner that when the observer was standing behind the patient the whole of the sole from the heel to the toe was seen at every step. The knee-jerks were entirely absent. There appeared to be some diminution in substance of the anterior tibial muscles when compared with his general nutrition, and they reacted only tardily to a strong faradic current. The weakness of the hands was extreme, on the left side the dynamometer registered only ten pounds, while on the right side the instrument marked thirty pounds, showing a serious diminution of power on this side also. Careful testing failed to find any alteration of tactile sensation in any of the paralyzed parts, but he stated that there had been some diminution of ordinary touch sense in the left hand.

The pupils reacted normally, and there was no weakness of vision. The optic disks were normal. Swallowing had not been affected, and was performed naturally at the time of observation. The voice was not, and had not been altered. The urine was normal. When directly questioned he said he had not had any sore throat, neither had there been any of an epidemic kind in the neighborhood in which he lived. There was no evidence of lead-poisoning or of any of the more generally recognized toxic conditions which produce peripheral palsies.

He was at once put upon a course of iron and arsenic, and gentle faradism applied to the limbs daily.

On February 3, 1888, the dynamometer registered thirty-eight pounds for the right hand, and twelve pounds for the left. He could undress himself, which he could not do before. The knee-jerk was still absent.

On March 2d the dynamometer registered seventy pounds for the right, and fifty pounds for the left hand. The knee-jerk was still absent.

On March 16th he was still improving. I failed to get the knee-jerk, though using the Jandrássik method, but the internal vastus contracted on direct percussion. The gait was quite natural.

On April 3d the right hand made seventy-five pounds, and the left seventy-two pounds, with the dynamometer. On percussion of the ligamentum patellæ a slight but distinct contraction of the right internal vastus was seen—once, on the left side, the foot was distinctly jerked forward.

On April 22d the knee-jerks could be easily elicited on both legs, and he was practically well.

The causal relations of this case are extremely interesting, though they do not here concern us, and the only point in the case to which I wish to direct attention is, that while the patient described various subjective disturbances of sensation, such as numbness, pricking, and the like, there was not, except in the left hand, at any time, any disturbance of the ordinary tactile sense. This is a seemingly paradoxical condition, which I have now and again observed in other conditions, which may have for their anatomical bases some changes in the peripheral nerves, such as "waking numbness," for instance.

Naturally, examples of pure motor or pure sensory neuritis will be found confined, for the most part, to the parenchymatous form of the disease, for, in the interstitial variety, it is obvious that there will be no respect for the kind of fibre involved, since the connective tissue is common to all parts of the compound nerve trunk. Perhaps the best available pathological evidence of the occurrence of a pure motor neuritis is that afforded by a remarkable case recorded by Joffroy.[1] This case, published in 1879, and frequently referred to by writers on peripheral neuritis, was one of peculiar interest, in which there supervened, during convalescence from smallpox a well-marked atrophy of the muscles with corresponding paralysis. During life the case had been regarded as one of poliomyelitis anterior acuta, the entire absence of any tactile sensory disturbance, in accordance with the views held in 1871, when the case was under observation, determining Joffroy to that diagnosis. Ultimately the patient died of tuberculosis, and the examination of the parts involved discovered no lesion of the spinal cord, or of the nerve-roots, but in the muscles and peripheral nerves the following changes were found:

> "The atrophied muscles presented a pale, dead-leaf color to the naked eye, and, on microscopic examination, disappearance of the striæ, proliferation of the intra- and extra-cellular nuclei and a granulo-fatty change of the substance of the muscle fibre. Several nerves from the limbs were examined. To the naked eye they presented no abnormal appearance. On microscopic examination, in all the sections examined, a number of perfectly healthy tubules were visible, but in a large number of other tubules there was noticed to be a segmentation more or less advanced of the white sheaths, and also numbers of fatty granules, which, in some instances, entirely filled the tubule, while the nuclei of the primitive sheath were largely increased in number."

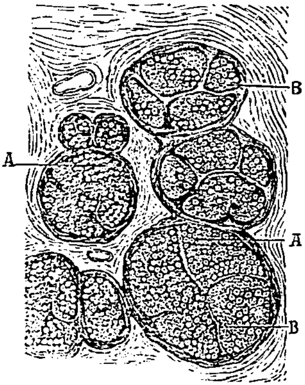
These appearances are clearly shown in the drawings accompanying Joffroy's paper—two of which are here reproduced.

The same description applies, also, to a second case, recorded in the same paper, in which a man suffering from phthisis was also paraplegic. The paralyzed limbs in this case showed normal sensibility to pain, temperature, and pressure, though, on account of mental hebetude, there was just the smallest reservation as to the integrity of the tactile sense to more delicate impressions.

---

[1] Archives de Physiologie Norm. et Path., vol. xi. p. 177.

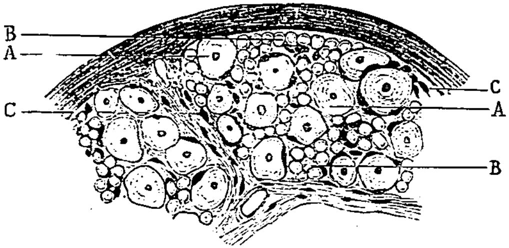
FIG. 2.



Section of the left radial nerve.

A. Normal nerve-fibres. B. Remains of nerve-fibres affected by parenchymatous change.

FIG. 3.



High power view of the same lesion.

A. Normal nerve-fibres. B. Remains of nerve-fibres affected by parenchymatous change.
C. Nuclei belonging to the primitive sheath.

In 1879, Déjerine[1] published an account of two cases of Landry's paralysis (a disorder which has, for one of its most striking, features, an absence of all disturbance of the tactile sensory function of the skin) in which there was found, post-mortem, a perfectly natural condition of the spinal cord, while there were most marked inflammatory changes in the anterior spinal roots, the nerve-trunks, and intra-muscular nerve-fibres.

In 1880 Eisenlohr[2] recorded a fatal case of lead palsy, in which, as is generally found, there was no affection of the tactile sense, but in which, at post-mortem, a well-marked neuritis of the affected parts was found.

Gombault[3] has also recorded a case of lead palsy under Charcot's care, in which were found changes in the peripheral nerves almost identical with those described in Joffroy's case above quoted. In this case there was, also, no disturbance of sensibility.

(The selective power of lead, or, if one may so speak, the striking originality of the distribution of lead palsy, is certainly most remarkable, for it appears to be able not only to take the motor and leave the sensory side of the nervous system, but seems to be still further able to confine its action to certain sections of the neuro-muscular apparatus. I need scarcely remark the great infrequency with which the muscles of the eye, the respiratory muscles, and the muscles of the face are affected in lead poisoning.)

Dr. Byrom Bramwell[4] has quite recently published a case of well-marked alcoholic paralysis, "in which myalgic pains and tenderness were absent, and in which there was very little affection of cutaneous (tactile) sensibility," "until," curiously, "the stage of convalescence was reached, when marked hyperæsthesia of the skin was developed."

All these cases seem to me to suggest that certain toxic substances, such as lead, and certain infectious processes, such as that of an ordinary septic wound, have, under some circumstances, the power of selecting certain parts of the nervous structures, according to the function of those structures, and not according to their anatomical position and association, for the field of their operation. This notion appears to me to be no more difficult of acceptation than is the fact that the influences which bring about such diseases as locomotor ataxy and a variety of spastic paraplegias pick out from the spinal cord a purely sensory tract in the one instance, and a purely motor tract in the other—both of which are simply conducting paths, taking on a diseased process at any point external to, and usually quite remote from, the central organs in which they end.

---

[1] Recherches sur les Lesions du Systeme nerveux dans la paralysie Ascendante Aigue. Paris, 1879.

[2] Deut. Arch. f. klin. Med., 1880, xxvi. p. 543, quoted by Dr. M. Allen Starr, in his Middleton-Goldsmith Lectures, Medical News, February 12, 1887.

[3] Archives de Physiologie Norm. et Path., 1873, p. 593.

[4] AMERICAN JOURNAL OF THE MEDICAL SCIENCES, June, 1884, p. 575.

In this regard I have often thought that rheumatic facial paralysis—that form of facial palsy, that is to say, which follows upon exposure to cold—is a condition of great interest. Of distinct and clear evidence of the part of the portio dura affected in such cases there is none that I know of forthcoming. The circumstances of the malady forbid that there should be such evidence. It is generally stated that the lesion in question is situated in the Fallopian canal, and that the suggestion of the palsy being due to the direct action of cold upon the terminals of the nerve is, for various reasons, untenable; still I fail to see why, such a cause as cold being allowed, it should be said to act upon a deeply seated and bone-protected part of the nerve-trunk in preference to those parts of it which are so much more obviously exposed on the face. However, adopting whichever hypothesis we may, I would ask, Why do we not see with something like corresponding frequency the sensory nerves of the same region affected by similar exposure? In my experience simultaneous affections of the corresponding fifth, in cases of Bell's palsy from cold, is exceedingly rare. May this not be that the exciting cause has some preference, as it were, for a motor, rather than for a sensory fibre?

I have ventured to treat the subject at such length because I deem it one of great practical importance. The more we can, if I may so speak, place the causes of paralysis in the periphery as distinguished from the centre of the nervous system, the more intelligible and the more successful will our treatment become. Moreover, it has seemed to me that there is a tendency to set up, while our experience is still limited, some affection of the tactile sensory nerves as a test of peripheral palsy, and so to exclude some cases from the category of peripheral neuritis to which they rightfully belong. It is against the institution of such a test that my remarks are directed.

THE PATHOLOGY AND TREATMENT OF INTRA-NASAL SCLEROSIS.

By Ralph W. Seiss, M.D.,
ADJUNCT PROFESSOR OF OTOLOGY AND INSTRUCTOR IN PATHOLOGY AND MICROSCOPY IN THE PHILADELPHIA POLYCLINIC.

The pathogenesis of advanced nasal catarrh has received but little attention from pathologists, and the descriptions of the conditions present are of the crudest character in all the general text-books.